Table 1. Questions asked of spouse and distribution of responses

| | |
|---|---|
| 1. What do you think when your partner does not show up at an appointed time? | |
| Sure that s/he is suffering from hypoglycaemia | 17.3 % |
| Concerned that something might have happened, e.g. accident | 60.3 % |
| Other thoughts | 22.4 % |
| 2. What is your emotional reaction to severe hypoglycaemia? | |
| Consternation | 67.3 % |
| Keep relatively calm | 31.7 % |
| 3. Is the potential of a severe hypoglycaemia a family burden? | |
| Always | 9.1 % |
| Sometimes | 47.3 % |
| Never | 41.8 % |

the possibility of severe hypoglycaemia was 'always' a burden.

Further research is needed to determine whether some spouses and marriages are more vulnerable to psychosocial problems secondary to recurrent severe hypoglycaemia. Clinical experience suggests that, for some couples, such hypoglycaemia stress can perpetuate problems with severe hypoglycaemia. For example, spousal concern about future severe hypoglycaemic episodes can trigger attempts to take more control over diabetes management, with more resistance to treatment assistance in patients.[8] Couples who demonstrate such power struggles should be referred for marital therapy to address these issues and improve their ability to cope with hypoglycaemia.

We would expect that spouses, like patients,[3] develop more fear of hypoglycaemia the more traumatic its past consequences. Severe hypoglycaemia may have similar psychosocial ramifications for children who have discovered their diabetic parents stuporous or unconscious and in need of emergency treatment. We believe that the psychosocial impact of hypoglycaemia on family members deserves increased clinical and empirical attention.

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**Panhypopituitarism, Neurosensory Deafness and Noonan's Syndrome in a Child of a Diabetic Mother: Role of Maternal Hypoglycaemia during Pregnancy in Induction of Congenital Lesions**

We describe a Type 1 diabetic patient with severe and recurrent hypoglycaemic episodes during her pregnancy who gave birth to a child with severe lesions. The patient is a 28-year-old woman with Type 1 diabetes mellitus diagnosed at the age of 3 years. Her knowledge of diabetes was good and, during the course of her disease, glycated haemoglobin measurements have usually been within or close to the reference levels. She never smoked. Her first two pregnancies and deliveries were without complications.

At the beginning of her third pregnancy at the age of 27 years she had no signs of nephropathy, normal blood pressure levels, and fundal photographs demonstrated only solitary dot haemorrhages. During the pregnancy she mesaured her blood glucose concentration 4–6 times daily. From the start of gestational week 6 and to the end of week 11, 41 of 120 registered blood glucose concentrations were $\leq 4$ mmol $l^{-1}$ and 19 $\leq 2$ mmol $l^{-1}$. $HbA_{1C}$ levels from week 6 to week 16 decreased from 6.3 % to 4.2 %, the lower reference level in the non-diabetic range in our laboratory. The levels were lower than in the previous two pregnancies. She suffered five hypoglycaemic comas, had marked tremor in connection with one hypoglycaemic episode and experienced pronounced tiredness during 13 further hypoglycaemic episodes. After 38 weeks of pregnancy she gave birth to a female infant with a weight of 3.250 kg, a length of 47 cm and head circumference of 38 cm. The infant cried at once, and the Apgar scores were 8, 8, 9, respectively. Her initial blood glucose concentration was low (1.4, 0.9, and 1.6 $mmol^{-1}$) and the hypoglycaemia lasted for 40 hours. In addition obstipation and gastric retention were found. Non-immunological icterus also developed. Further investigations revealed panhypopituitarism in the infant. Computer tomography of the brain and pituitary demonstrated no pathological morphology. l-Thyroxine, cortisol, and growth hormone replacement was started at day 10 postnatally. Later she required hearing aids for congenital neurosensory defects and spectacles because of myopia. At the age of 5 years, she has moderate psycho-motor retardation and has been diagnosed with Noonan's syndrome.[1] The diagnosis is based on the findings of short neck stature, hypertelorism, characteristic facial changes, mild mental retardation, and peripheral pulmonary stenosis.

Noonan's syndrome can occur spontaneously, occasionally in association with pituitary hormonal deficiencies, and



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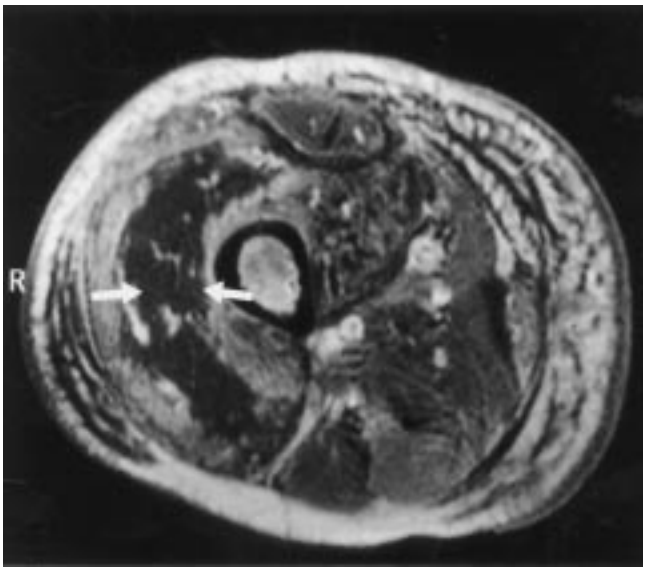


Figure 1. Transverse T1-weighted SE image obtained after intravenous injection of contrast material shows a central dark, non-enhanced area inside the right enlarged quadriceps muscle with surrounding enhancement. Linear enhanced signal infiltrates (arrows) are present through the dark core and tissue planes are preserved. R=right

could arise coincidentally in the baby of a mother with diabetes.

In this particular case, the mother had experienced recurrent severe hypoglycaemia during early pregnancy which could have been of importance.

The increase in perinatal morbidity and mortality in diabetic pregnancy is well known and has been shown to be associated with maternal hyperglycaemia.[2] Evidence that maternal hypoglycaemia may cause human fetal lesions is scarce. In a review of eight pregnant women who were subjected to insulin-induced hypoglycaemic comas for treatment of psychiatric diseases it was found that two delivered normal children, four macerated fetuses, and two mentally retarded children of whom one had skeletal skull anomalies and atrophy of the optic nerve.[3] In animal experimentation, pharmacological doses of insulin administered to pregnant animals were associated with fetal structural damage and increased fetal resorption rate.[4,5] Preventing the hypoglycaemia in the insulin-treated rats normalized embryonic development and *in vitro* addition of pharmacological doses of insulin to normoglycaemic serum did not cause malformations in the cultured embryos.[6]

These data do raise the possibility that severe recurrent hypoglycaemia may be toxic to fetal development.

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**Diabetic Muscle Infarction: a Difficult Diagnosis Suggested by Magnetic Resonance Imaging**

We report the case of a 22-year-old woman with Type 1 diabetes mellitus of 6 years' duration and anorexia nervosa who was admitted to our unit because of hypoglycaemic coma. She had a bedsore of the right buttock, infected by enterococcus faecalis. Her diabetes was poorly controlled ($HbA_{1c}$ 10 %) and was complicated by cardiac and gastrointestinal autonomic neuropathy, cardiac failure, and proliferative retinopathy. One morning, the patient awoke with pain in the right thigh which was swollen (maximal circumference of 41 cm, versus 38 cm on the left side). The calves were painless and Homans's sign was absent. The laboratory tests showed a C-reactive protein of 33 mg $dl^{-1}$ and a white-cell count of 12 000 $mm^{-3}$. Creatine kinase levels were normal. The Doppler study of the right leg was normal. The pain worsened and a first MRI study of the right thigh suggested soft tissue infection on the basis of the hypersignal of the quadriceps muscle on T2-weighted images and diffuse enhancement on post-contrast T1 weighted images with central and focal dark areas. The evolution worsened and a second MRI scan revealed significant enlargement of the muscle with the same characteristics on T2-weighted images. On T1-weighted images after gadolinium injection (Figure 1), the unhanced central core within the muscle appeared larger with a surrounding enhanced rim, mimicking a muscular abscess. However, linear enhanced signal infiltrates were present through the dark core. On clinical, biological and radiological basis, a pyomyositis was suspected and surgical intervention occurred. The surgeon did not find an abscess but necrotic tissue. The histologic examination showed necrotic myocytes without nuclei. The perimysium contained red cells. There was no evidence of vasculitis or microvessel angiopathy, and bacteriologic samples were negative.

Skeletal muscle infarction, firstly described in diabetes in 1965[1] is a rare complication of diabetes and may be misdiagnosed as a neoplasm, an abscess or myositis, as recently reported.[2] It occurs in patients with poorly controlled diabetes and with diabetic complications, as in our patient. The infarction has been suggested to result from ischaemia caused by arteriosclerosis and microangiopathy, although we did not find histological evidence of either in the present case. Creatine kinase levels may be normal or slightly elevated.[2] Our case illustrates that this difficult diagnosis must be considered in the face of a painful and tender swelling of a leg occurring abruptly in a diabetic patient. We also suggest that post-contrast T1-weighted MRI revealing the presence of enhanced linear signal infiltrates through an abscess-like lesion preserving the tissue planes may suggest diabetic infarction.

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